ASSESSMENT OF LEFT VENTRICULAR FUNCTION IN ISCHAEMIC STROKE

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DECLARATION

I solemnly declare that this Dissertation entitled "Assessment of Left Ventricular Function In Ischeamic stroke Patients of Government General Hospital and Madras Medical College, Chennai" was done by me at Madras Medical College and Government General Hospital during 2004 –2007 under the guidance and supervision of **Prof. Dr. V. RAJI**. This dissertation is submitted to the Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of **M.D., Degree in General Medicine, Branch- I.**

Place :

Date :

Dr. P. MALARVIZHI

CERTIFICATE

This is to certify that the Dissertation entitled "Assessment of Left Ventricular Function In Ischeamic stroke Patients of Government General Hospital and Madras Medical College, Chennai" is a bonafide work done by Dr. P. Malarvizhi at Madras Medical College, Chennai in partial fulfillment of the University rules and regulations for award of M.D., Degree in General Medicine under my guidance and supervision during the academic period from May, 2004-2007.

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CONTENTS

ABBREVIATIONS

BP	Blood Pressure
BBB	Bundle branch block
CXR	Chest X Ray
CAD	Coronary Artery Disease
CVS	Cardio Vascular System
CNS	Central Nervous System
CHG	Complete Hemogram
DDF	Diastolic Dysfunction
DM	Diabetes Mellitus
DOA	Date of Admission
DOD	Date of Discharge
ECG	Electro Cardiography
EF	Ejection Fraction
ECHO	Echo Cardiogram
GGH	Government General Hospital
H/O	History of
i-e	That is
LV	Left Ventricular
LVSD	Left Ventricular Systolic Dysfunction
LVH	Left Ventricular Hypertrophy
MI	Myocardial Infarction
NYHA	New York Heart Association
NOMAS	Northern Manhattan Study
PVD	Peripheral Vascular Attack
TIA	Transient Ischaemic Attack
2 – D	2 Dimensional

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INTRODUCTION

Cardiac disease is a major risk factor for stroke, ranking third after age and hypertension.[1] Congestive Cardiac failure ranks second in cardiogenic stroke risk [1], with a two fold to three fold relative risk[1,2]. The prevalence of cardiac failure increases substantially in prevalence as the population ages. Cardiac failure is also associated with high mortality, with a 15 year total mortality rate estimated at 39% for women and 72% for men[3].

LVSD is common and treatable, accounting for 8%of people aged 25-75 years and 12% of 45-75 years. Of the 8% ,4% are asymptomatic[6]. The patients who appear to be at high risk of LVSD are those with ischaemic heart disease, hypertension or diabetes, and smokers[9-11]. However, echocardiographic screening of all hypertensives, all diabetics and all smoker for LVSD would be a daunting and costly process. A more cost- effective approach might be to wait for the first presentation of a vascular episode, and to perform routine echo screening at that time. The first vascular episode could be either a myocardial infarction (MI), a transient ischaemic attack (TIA), a cerebro vascular accident or peripheral vascular disease. In normal clinical practice , MI patients are now a days screened for LVSD during their hospital admission, but, patients who have had CVA/ TIA/ PVD are not routinely screened for LVSD . Yet, the presentation with one vascular episode in the form of stroke could be a golden opportunity to detect and treat LVSD, and thereby reduce the subsequent incidence of overt heart failure, and perhaps even sudden cardiac death.

We are aware that LVDD is also a major risk factor contributing towards mortality especially in hypertensives and elders. It is in this context,

I have tried to assess LV function in patients who presented with atherothrombotic stroke.

REVIEW OF LITERATURE

DEFINITION

Stroke or cerebro vascular accident is a rapidly developing clinical symptom and /or signs of focal, and at times global (applied to patients in deep coma and to those with sub arachnoid haemorrhage) loss of cerebral function with symptoms lasting more than 48 hrs or leading to death with no apparent cause other than that of vascular origin.

Cerebral infarction accounts for approximately 80% of the stroke as opposed to 10% due to primary intracerebral bleed, 5% due to subarachnoid haemorrhage and another 5% of uncertain etiology[4].

ATHEROSCLEROSIS AND STROKE

Stroke can affect a person at any age. Still, advancing age is the single most important factor that predicts an increased likelihood of ischemic stroke. Risks increase rapidly after the age of 55 in both men and women of all ethnic groups[12-15]. The risks of stroke approximately double with every 10-years increase in age. Ischemic stroke is a leading cause of acute- onset neurological impairments among persons older than 55, and stroke should be a leading consideration to explain new neurological symptoms in this population. Atherosclerosis and ischemic heart disease are the primary substrates for ischemic stroke in this age group. The formation of atherosclerotic plaques and the development of arterial stenoses increase with advancing age[16,17]. Still, the influence of well –recognized risk factors

(hypertension, smoking, etc.) on the development of advanced atherosclerotic lesions is not as great in the elderly as among young persons.[16] Some of the lack of an association between the traditional atherosclerotic risk factors and stroke in older persons with the highest risk.

HYPERTENSION AND STROKE

Hypertension is calculated to be a factor in 70% of strokes and among survivors, it identifies a patient as having an increased risk of a second event[18-21]. The risk of stroke increases 10-12 times if diastolic blood pressure is 105mm Hg in comparison to a normal diastolic blood pressure of approximately 76mm Hg[8,22]. Overall, arterial hypertension increases the likelihood of stroke. The risk rises rapidly with higher levels of blood pressure[22]. The impact of an increase in diastolic blood pressure is much greater in young adults than in the elderly. Although elevated diastolic blood pressure is a usual marker for hypertension, isolated systolic hypertension also predicts stroke in elderly persons[22,23]. Isolated systolic hypertension also is correlated with increased thickness and plaque formation of the internal carotid artery. Both elevated diastolic and systolic blood pressure are associated with increased concentrations of hemoglobin, which is a risk factor for ischemic stroke[24].

Chronic hypertension promotes the development of both large and medium caliber artery atherosclerosis and the lipohyalinosis of small penetrating arteries of the brain[25]. The vascular endothelium is the central focus of the effects of hypertension that promotes turbulence at the site of an

atherosclerotic plaque. Because hypertension leads to coronary artery disease, cardiomyopathy, and atrial fibrillation, it is an indirect risk factor for stroke secondary to cardioembolism. Reducing diastolic blood pressure by approximately 5mm Hg can reduce the incidence of an mortality from stroke by 42% and 30% respectively[26,27]. In elderly patients, a drop of systolic blood pressure by approximately 11mm Hg can lead to a 30% decline in the frequency of stroke. A report from Asia concluded that a population-wide reduction of 3mm Hg in diastolic blood pressure could reduce the number of strokes by one-third[28]. The optimal levels for blood pressure are 80-85mm Hg diastolic and 130-140mm HG systolic.

DIABETES MELLITUS AND STROKE

Persons with either type I or type II diabetes mellitus have an increased susceptibility for larger artery atherosclerosis and small artery occlusive disease. Although glucose intolerance is accompanied by arterial disease, it may not be associated with an increased risk of stroke. In contrast, diabetic patients often have elevated levels of triglycerides or high-density lipoproteins that persist even with control of the hyperglycemia[29]. Diabetes mellitus also leads to renal or cardiac disease, which indirectly promote arterial hypertension and stroke. Autonomic disturbances secondary to diabetic neuropathy also may play a role by causing hypotension . Diabetes also increases level of fibrinogen; and clotting factors increase platelet aggregation, which in turn promotes arterial thrombosis.

ELEVATED BLOOD LIPIDS AND STROKE

The incidence of coronary artery disease and atherosclerosis of major intracranial or extracranial arteries increases with rising levels of serum cholesterol. Recent data show that hyper-cholesterolemia is a key risk factor for ischemic stroke, especially in men under the age of 60. Hyperlipidemia causes a modest increase in risk of stroke in persons older than 62. Some of the risk may be indirect, due to the potent effects of hyperlipidemia on coronary artery disease, leading to myocardial infarction and cardioembolism. Sequential duplex ultrasound studies of the carotid arteries demonstrate that hypercholesterolemia accelerates the course of early atherosclerotic lesions (intimal-medial thickness [IMT]) [30].

HEART DISEASE AND STROKE

Heart disease is a leading potential cause of ischemic neurological symptoms. In addition to causing ischemic stroke secondary to cardioembolism, heart disease also produces recurrent neurological complaints that are included in the differential diagnosis of transient ischemic attack (TIA). Intermittent cardiac arrhythmias, most commonly intermittent complete heart block or the bradycardia - tachycardia (sick sinus syndrome), are frequent cardiac cause of recurrent syncope in the elderly. Cardioembolism accounts for approximately 20% - 25% of ischemic strokes and is a leading cause of stroke in persons of all ages. Emboli from the heart can go to any intracranial artery. Both proximal and distal intracranial arterial occlusions can develop. As a result, cardioembolism

should be considered in the differential diagnosis for cause of symptoms in any patient with a TIA or an ischemic stroke in either the carotid or the vertebrobasilar circulation[31,32]. The types of heart disease leading to stroke also varies by age; coronary artery disease and atrial fibrillation are leading etiologies in persons older than 45, whereas valvular diseases are more prominent in younger persons. Because symptomatic coronary artery disease appears at earlier ages in men, it is a leading substrate for cardioembolic stroke in middle-age men. Conversely, atrial fibrillation is a leading cause of stroke in women, especially in those older than 75. Most patients with cardioembolic stroke have a prior history of heart disease. Still, stroke can be a first presentation of otherwise occult heart disease. In general, patients have cardiac symptoms, including chest pain, fatigue, or dyspnea, and clinical evidence of heart disease, such as a murmur, cardiomegaly, cyanosis, or peripheral edema. Patients also may have been treated with cardiovascular medications or cardiac procedures. A past history of an abnormal chest x-ray or electrocardiogram also might suggest structural heart disease.

CARDIAC DYSRHYTHMIAS AND STROKE

The two abnormalities of cardiac rhythm most often associated with focal cerebral ischemia are atrial fibrillation and the sick sinus syndrome.

Diffuse nonfocal cerebral ischemia (i.e., syncope or Stokes- Adams attacks) may be caused by cardiac dysrhythmias that induce cerebral hypoperfusion. Reduction in cerebral perfusion is usually the result of

complete heart block or paroxysmal tachyarrhythmia associated with a rapid ventricular rate of more than 180 beats per minute (e.g., paroxysmal atrial tachycardia, ventricular tachycardia, or ventricular fibrillation). Focal cerebral ischemia is rare with such distrubances of cardiac rhythm.

ATRIAL FIBRILLATION AND STROKE

Nonvalvular atrial fibrillation (NVAF) is the term commonly used to specify the group of patients in which the underlying cause is other than valvular disease. NVAF affects 2% to 5% of the general population over the age of 60. Atrial fibrillation is the most common cardiac abnormality correlated with embolic stroke. The arrhythmia appears to predispose thromboembolism by reducing the contraction of the left atrium, decreasing flow in the left atrial appendage, increasing turbulence in the left atrium, and leading to enlargement of the left atrium and left atrial appendage[33.] All of these factors promote stasis and the formation of clots with in the left atrium. It is not clear whether atrial fibrillation is the cause or a complication of dilation of the left atrium. The risk of embolism in patients with the arrhythmia is increased by the presence of structural heart disease. Conversely, the risk of embolism from structural heart disease is increased by the presence of atrial fibrillation. For example, the presence of atrial fibrillation after myocardial infarction greatly increases the risk of stroke. The finding of atrial fibrillation is associated with an increase in the risk of stroke by a factor of 5-7 times that found in persons with sinus rhythm[58]. In persons who have heart disease not complicated by atrial

fibrillation, the risk of stroke is approximately 6% per year. The presence of arrhythmia increases this risk to approximately 15% per year.

Atrial fibrillation is more common in persons who have a history of hypertension, diabetes mellitus, and smoking, even if they do not have symptoms of heart disease. Afrial fibrillation is also more common in persons with a history of congestive heart failure[34]. Atrial fibrillation complicates several heart deseases, and a majority of patients with atrial fibrillation have an underlying cardiac abnormality. With the decline in the prevalence of rheumatic heart disease secondary to improved antibiotic treatment, coronary artery disease and hypertension are the most common heart disease associated with atrial fibrillation.

ACUTE MYOCARDIAL INFARCTION AND STROKE

Ischemic stroke is a leading non-cardiac complication of acute myocardial infarction. Some patients, especially diabetics, have relatively little pain at the time of myocardial infarction, and the acute cardiac event is not recognized. Thus, embolic stroke may be a presenting symptom. The risk of stroke following myocardial infarction probably has been reduced by the widespread administration of thrombolytic, antithrombotic, and antiplatelet aggregating medications to treat acute myocardial ischemia[35,36]. The risk of stroke in persons with acute myocardial infarction appears to range from 1% to 6%. The occurrence of a neurological event is associated with an increased risk of death. In part, the increased mortality is secondary to the brain injury. It also reflects the serious ventricular injury

that leads to thromboembolism. A strong time relationship for embolic complications is present. The first days and weeks after myocardial infarction are the period of highest risk for embolism. Moore et al[37] calculated that the daily risk for stroke was approximately 9/10,000 during the first 28 days after myocardial infarction. This interaction should not be surprising because the first few days after the acute heart injury are when cardiac instability, fresh myocardial injury, and changes in coagulation are most likely. By 6 months after the cardiac event, the likelihood of a complicating stroke is relatively low. This time interval is important when making recommendations about anticoagulant treatment to prevent cardioembolic stroke after myocardial infarction.

The risk of stroke after myocardial infarction is increased with an anterior myocardial infarction, a history of previous myocardial infarction, hypertension, atrial fibrillation, or age greater than 70. The risk in persons with non-anterior myocardial infarction is less than 1% whereas anterior injuries are associated with a risk of approximately 2% -6%. Echocardiographic detection of an intraventricular or mural thrombus is associated with a markedly increased risk of embolization. The thrombus usually is adjacent to a hypokinetic or akinetic segment of the left ventricle. Martin and Bogousslavesky[38] noted that all patients with stroke after myocardial infarction had evidence of an akinetic segment. Left ventricular thrombi are rare in patients with inferior myocardial infarctions, whereas up to 40% of patients with anterior infarctions have clots detected by imaging tests. Embolism also is more likely in persons who have atrial

fibrillation, congestive heart failure, or poor left ventricular function complicating the myocardial injury.

Anticoagulation is a key component of the regimen to prevent cardioembolic stroke after myocardial infarction[39]. Turpie et al reported that heparin therapy lowered the frequency of mural thrombi from 32% to 11% in a group of high-risk patients, reducing the risk by a ratio of 0.46-0.60 (0.30-0.90) [40]. Most patients are treated with anticoagulants, antiplatelet aggregating agents, and thrombolytic agents during the first hours after myocardial infarction. Both anticoagulants and thrombolytic agents appear to prevent formation of a mural thrombosis, but evidence that antiplatelet aggregating agents are effective is lacking. The key to stroke prevention is to identify the highest risk patients and continue anticoagulation for at least 6 months. The desired level of anticoagulation is an INR of 2-3.

STROKE IN PATIENTS WITH IMPAIRED LEFT VENTRICULAR FUNCTION

The relative risk of stroke in patients associated with congestive heart failure is about 4.1 among those 50-59 yrs of age[1,2]. The risk of stroke associated with cardiac failure is slightly higher among men than women, but impaired left ventricular function may be a more powerful risk factor for stroke in women, reflecting the sex related difference in the nature of underlying cardio vascular disease[41].

EF AND HEART FAILURE

EF is the proportion of left ventricular volume emptied during systole. It is a reliable measure of left ventricular systolic dysfunction that can be assessed non invasively by echocardiography the normal value is 50% to 70% when left ventricular systolic function is impaired, EF declines unless there is a corresponding reduction in workload, as ocurs in states of peripheral vasodilatation or valvular regurgitation. Consequences of low EF include elevated left vertricular filling pressure and a fall in stroke volume that reduces the systemic blood flow. Peripheral vasoconstriction often develops to a degree beyond that required to compensate for the fall in cardiac output and maintain blood pressure. Further more, the reduced stroke volume creates a condition of relative stasis with in the left ventricle that may activate coagulation processes and increase the risk of thromboembolic events. There is a significant risk of thromboembolic events for every 10% decrease in EF among population. The recurrent stroke rate is much higher than the rate of first-ever stroke in patients with cardiac failure.

Roughly half of patients with symptomatic heart failure in the community have reduced systolic function, and half have preserved function. The epidemiology of symptomatic heart failure with reduced systolic function differs from that of heart failure with preserved systolic function; patients with preserved function are, on average, older, higher proportions are women or have comorbidity, and they have better age-

adjusted survival. The causes of heart failure in patients with preserved systolic function also differ from those with reduced systolic function.

About half of cases of left-ventricular systolic dysfunction are asymptomatic, which raises questions about screening for symptomless cases. The syndrome of heart failure arises as a consequence of an abnormality in cardiac structure, function, rhythm, or conduction. In more developed countries, ventricular dysfunction is the commonest underlying problem. It results mainly from myocardial infarction (systolic dysfunction), hypertension (diastolic and systolic dysfunction), or in many cases both. Other common causes include "idiopathic" dilated cardiomyopathy, some cases of which could have a genetic basis, and alcoholic cardiomyopathy.

VENTRICULAR REMODELING IN HEART FAILURE

Increased level of circulating neurohormones are only part of the response seen after an initial insult to the myocardium. Left ventricular remodeling is the process by which mechanical, neurohormonal, and possibly genetic factors alter ventricular size, shape, and function. Remodeling occurs in several clinical conditions, myocardial infarction, cardiomyopathy, hypertension, and valvular heart disease; its hallmarks include hypertrophy, loss of myocytes, and increased interstitial fibrosis[42].

For example, after a myocardial infarction, the acute loss of myocardial cells results in abnormal loading conditions that involve not

only the border zone of the infarction, but also remote myocardium. These abnormal loading conditions induce dilatation and change the shape of the ventricle, rendering it more spherical, as well as causing hypertrophy. Remodeling continues for months after the initial insult, and the eventual change in the shape[43] of the ventricle becomes deleterious to the overall function of the heart as a pump.

Several trials involving patients who were studied after a myocardial infarction or who had dilated cardiomyopathy found a benefit from ACE inhibitors, beta-adrenergic antagonists, or cardiac resynchronization. Such beneficial effects were associated with so-called reverse remodeling, in which the therapy promoted a return to a more normal ventricular size and shape. The reverse -remodeling process is a mechanism through which a variety of treatments palliate the heart - failure syndrome.

The myocardial conduction system is vulnerable to the same pathophysiological processes that occur in the myocytes and interstitium, with altered conduction properties observed in response to ischemia, inflammation, fibrosis, and aging. Supraventricular arrhythmias, particularly atrial fibrillation, are often the precipitating events that herald the onset of either systolic or diastolic heart failure. Elevated ventricular end-diastolic pressure in a patient with hypertension or abnormal myocardial function leads to atrial stretch, which in tun incites electrical instability. Recognition of the presence of atrial fibrillation in a patient is critical, since several studies have now demonstrated the effectiveness of oral anticoagulant therapy for the prevention of stroke[44].

Abnormal myocardial conduction can also lead to delays in ventricular conduction and bundle-branch block. Left bundle- branch block is a significant predictor of sudden death and a common finding in patients with myocardial failure. Its presence also affects the mechanical events of the cardiac cycle by causing abnormal ventricular activation and contraction, ventricular dyssynchrony, delayed opening and closure of the mitral and aortic valves, and abnormal diastolic function. Hemodynamic sequelae include a reduced ejection fraction, decreased cardiac output and arterial pressure, paradoxical septal motion, increased left ventricular volume, and mitral regurgitation[45]. Ventricular arrhythmias are thought to be secondary to a dispersion of normal conduction through nonhomogeneous myocardial tissue, which promotes repetitive ventricular arrhythmias.

AIM OF THE STUDY

To study and evaluate left ventricular function in patients with ischaemic stroke.

To analyse whether LV dysfunction could be a surrogate marker for in hospital mortality in patients developing ischaemic stroke.

MATERIALS AND METHODS

STUDY DESIGN

Randomised prospective observational study.

SETTING

Medical wards of Government General Hospital, Chennai.

Study group : 142 patients diagnosed as ischaemic stroke.

Period of study : April 2005 to September 2006.

LV function was assessed by trans thoracic 2 - dimensional echo cardiography in patients admitted with ischaemic stroke under various medical units of our government general hospital

EXCLUSION CRITERIA

- Patients under the age of 39 yrs were excluded from the study group.
- Patients with haemorrhagic stroke were excluded from the study group.
- Patients with valvular heart disease were excluded from the study group.

All the study group patients underwent transthoracic echocardiopgraphy as a part of the study.

All patients had a thorough clinical , neurlogical examination with careful evaluation of history. Importance was given to symptoms and signs of cardiac disease in addition to neurological findings.

Routine laboratory tests included urine analysis, complete blood counts, serum electrolytes, blood glucose and serum cholesterol determination. Arterial hypertension was defined as presence of a positive history of antihypertensive treatment or blood pressure values >140 /90 mmhg on admission. Hypercholesterolemia was defined as a total serum cholesterol >240mg /dl. or presence of appropriate drug treatment earlier. Diabetes mellitus was defined based on abnormal fasting glucose >125mg /dl, positive history or presence of oral hypoglycemic agents intake or insulin treatment. Coronary artery disease included history of myocardial infarction or typical angina or the patients reporting of a positive diagnostic test (stress test, Coronary angiography) or drug treatment, 12 lead ECG and CXR were taken for all the patients . The neurological work up included head CT and the patients showing haemorrhage on the CT were excluded from the study group[46].

ECHOCARDIOGRAPHIC EVALUATION

Transthoracic two dimensional echocardiography was performed in all study patients.

LVEF was measured by the following formula

$$\frac{LVEDV - LVESV}{LVEDV}$$

LVEDV = LV end diastolic volume

LVESV = LV end systolic volume

LVEF was then categorised as

Normal = >50%

Mildly decreased = 41-50%

Moderately decreased = 31-40%

Severely decreased = 30% or less

LV diastolic dysfunction was graded as[47,48]

- Normal
- Grade I (delayed relaxation)
- Grade II (Pseudo normal filling)
- Grade III (restrictive filling) based on mitral flow velocities and filling times.

LVH was defined as[49]

Increase in interventricular septal thickness and posterior wall thickness beyond 1.1cm during diastole as excess by M-mode LV

measurements in parasternal long axis and apical views in left lateral position.

LV mass was derived using the formula described by Devereux and associates[50]

LV mass (grams)= $0.80 \times 1.04 [(VSTd+LVIDd+ PWTd)^3-(LVIDd)^3]+0.6$

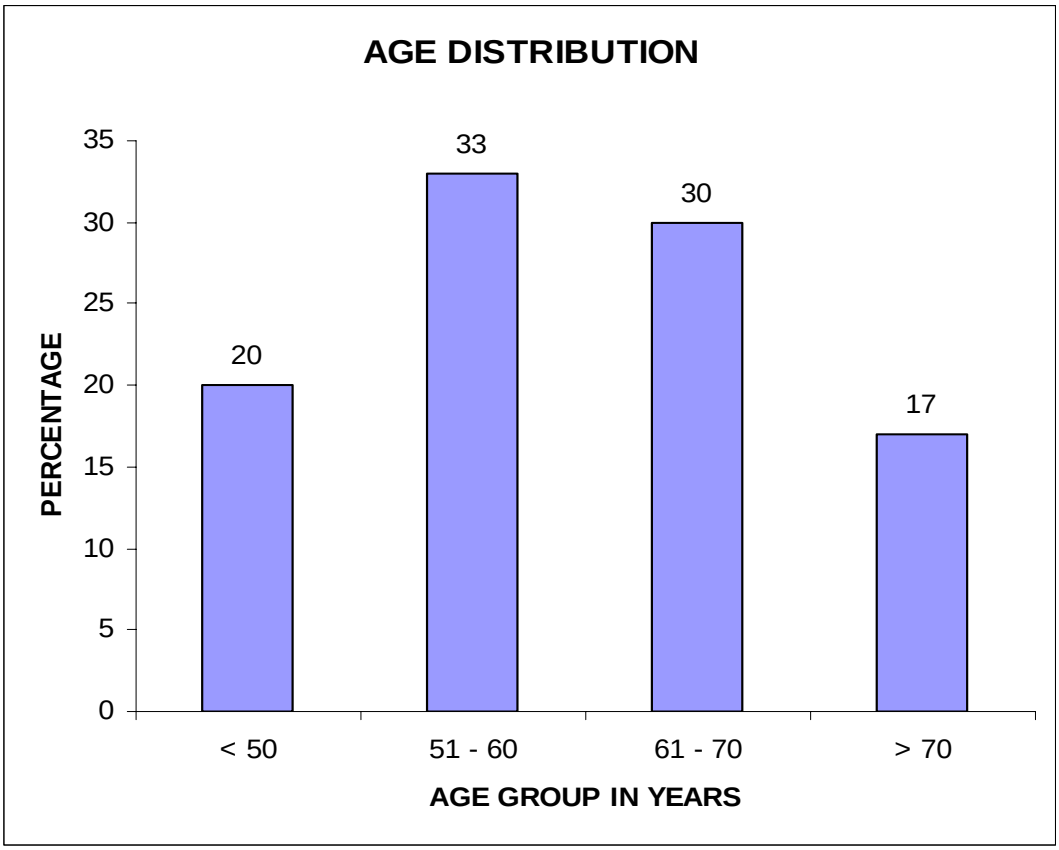
- VSTd is ventricular septal thickness at end diastole
- LVIDd is LV internal dimension at end diastole
- PWTd is LV posterior wall thickness at end diastole.

LV mass was corrected for height $^{2.7}$ (LVMI), and expressed in units of grams/meter ($g/m^{2.7}$). The presence of left ventricular hypertrophy was defined for LVMI $>51 g/m^{2.7}$ in either gender.

RESULTS

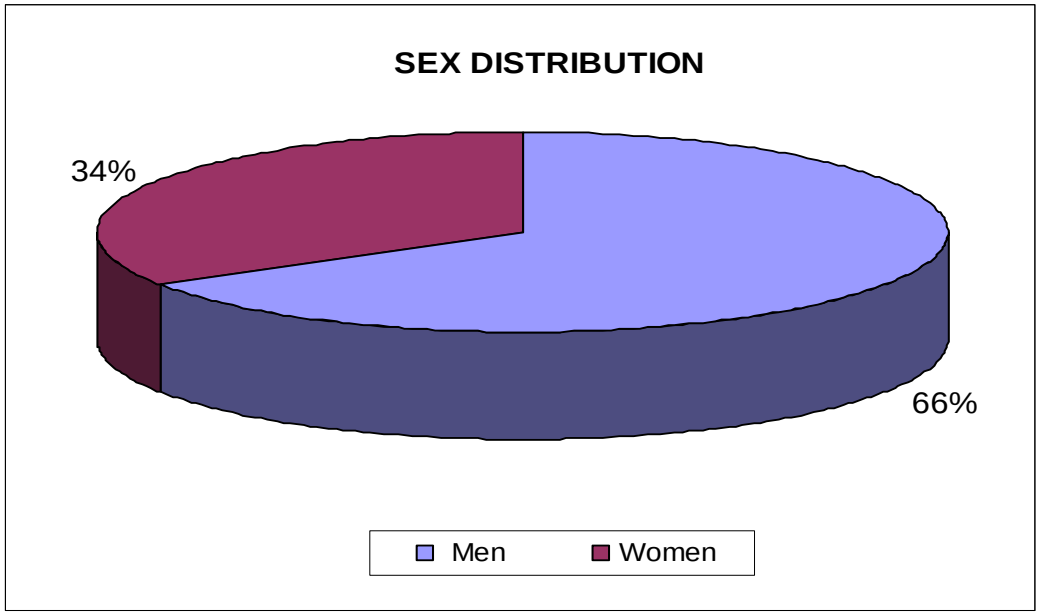
AGE

142 Patients of the study group were divided into various sub groups. Ischaemic stroke was most commonly observed between 51-60 years of age followed by patients aged 61-70 years .The mean age of the patients was `58 years.

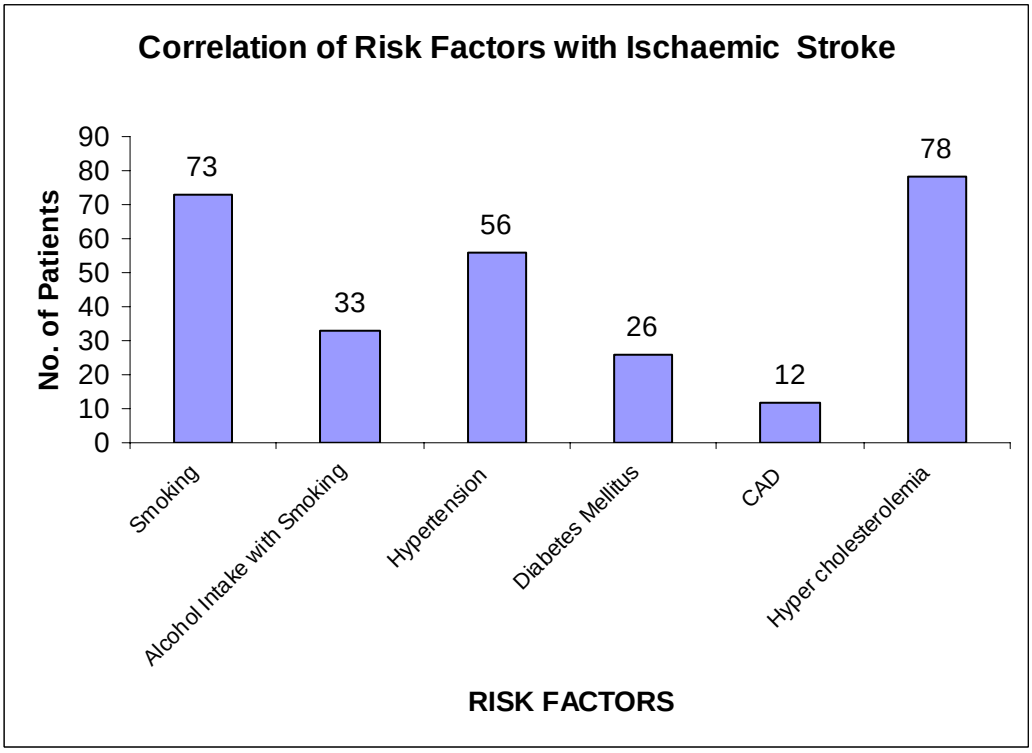


SEX

Most of the patients were men (66 %) as against women (34%)



RISK FACTOR PROFILE



Smoking

Smoking was one of the most common risk factor observed in 73 patients.

All of these patients were men. Out of 94 men, 73 patients were chronic smokers (51%). The mean age of the smokers was 54 years. Associated history of chronic alcohol intake was seen in 33 male patients. Associated hypertension was seen in 11 patients which had statistical significance (P- value .00). Coexisting diabetes mellitus was observed in 8 patients. This correlation had a statistical significance (P- value .01) Prior history of coronary artery disease was obtained in 6 patients. This was not statistically significant. Hypercholesterolemia was noticed in 42 patients. Past history of stroke was present in 25 patients. History of shortness of breath of varying degree suggestive of left heart failure was observed in 20 patients.

History of angina and palpitation was observed in 13 patients. There is no statistical significance associated with the heart failure symptoms among the smokers. 33 of the 73 patients had ECG changes of varying degree out of which 12 patients had ECG changes suggestive of left ventricular hypertrophy. 7 patients had ECG Changes of previous of myocardial infarction. 13 patients showed changes in the chest x- ray. 8 patients had cardiomegaly in the chest x- ray . Echocardiographically LVH was evident in 34 patients. E diastolic dysfunction was present in 5 patients. Clinical picture of stroke w as severe in smokers associated with altered mentation, dense hemiplegia and massive infarct on the C.T . This association had

statistical significance (P= value .00). In hospital mortality was observed in 3 patients .

Alcohol

Alcohol intake was observed in 33 of the 94 men (23%). All of them were men. They had associated history of chronic smoking. 21 patients had associated hypercholesterolemia. 4 patients had diabetes mellitus and 4 patients had coronary artery disease. Shortness of breath on exertion was observed in 7 patients. History of angina, palpitation was present in 6 patients. Chest skiagram showed cardiomegaly in 1 patient. Echographically L.V.H was evident in 13 patients. L.V. systolic dysfunction was present in 6 patients. Diastolic dysfunction was present in 3 patients. One patient had in hospital mortality.

Hypercholesterolemia

78 of the 142 patients had hypercholesterolemia (54%). Out of which 58 were men and 20 were women. It was the most common risk factor observed among the study group patients. It was most commonly observed in the age group between 51-60 years followed by 61-70 years. The mean range of blood cholesterol was between 240-260 mgms%. 14 patients had associated diabetes mellitus. 25 patients had associated hypertension. Hypertension had statistically significant association with hypercholesterolemia. (P= value .04). 5 patients had past history of coronary artery disease .

Previous history of stroke was present in 24 patients. Echocardiographically L.V.H was seen in 29 patients. 14 patients had left ventricular systolic dysfunction. 8 patients had left ventricular diastolic dysfunction. C.T scan showed massive infarct in 35 patients. This had statistical significance (P- value- .02). 7 patients had mortality in the hospital.

Hypertension

Hypertension was observed in 56 of the 142 patients studied (39.4%). It was most commonly observed in the age group between 50 – 70 years when compared to those below 50 years and more than 70 years of age group. It was more frequent in women compared to men in the ratio of 36:20

17 patients had history of angina and palpitation. History of shortness of breath was observed in hypertensive patients which had no statistical significance.

Co existing diabetes mellitus was seen in 6 patients. Co existing hypercholesterolemia was observed in 25 patients. 5 patients had associated coronary artery disease (P- value .02).37 patients had E.C.G changes .

Echo cardiographically LVH was evident in 29 patients. 40 out of 56 patients had normal LV systolic function 16 patients had LV systolic dysfunction. 5 patients had LV diastolic dysfunction . 5 patients had mortality during the hospital stay.

Diabetes Mellitus

26 of the 142 patients had diabetes mellitus (18%). It was observed as a risk factor equally in both sexes (P= value .05). Previous history of coronary artery disease was present in 12 patients. Coexisting C.A.D was more commonly seen in men compared to women . Past history of stroke was present in 8 patients. History suggestive of angina , palpitation and shortness of breath was observed among the diabetic spatients which had no statistical significance. ECG changes of previous myocardial infarction was seen in 6 patients. This was associated with statistical significance (P= value .03). Chest X-ray showed cardiomegaly in 8 patients with statistical significance (P= value .02). 14 patients had left ventricular systolic dysfunction echocardiographically. Two patients had left ventricular diastolic dysfunction. 1 patients had LVH. Two patients had in hospital stay mortality with statistical significance (p value .00).

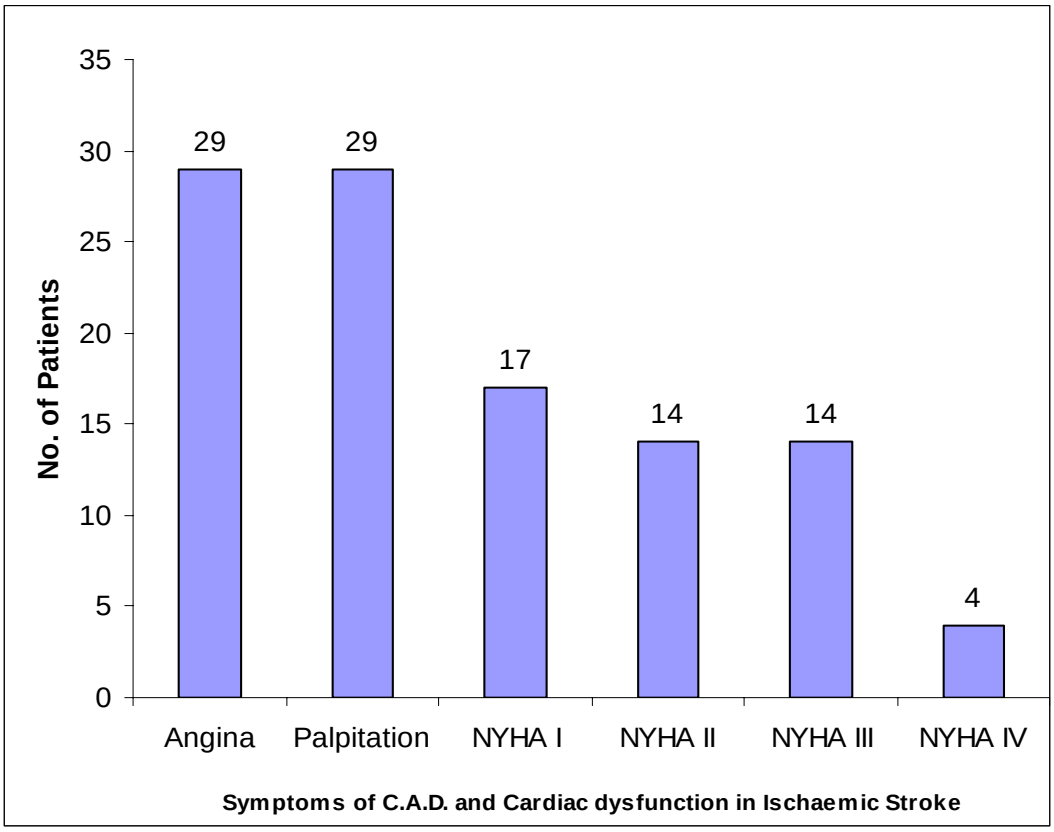
Coronary Artery Disease

History of coronary artery disease was present in 12 patients (8.4%) C.A.D w as more frequent in men when compared to women in the ratio of 8:4. Most common age group with C.A.D was between 51-60 years. History of breathlessness on exertion of NYHA class III was more frequently observed in C.A.D patients. This association had statistical significance (P= value .00). Past history of stroke was obtained in 3 patients. Associated diabetes mellitus was present in 12 patients. Hypertension was seen in 5 patients. Elevated cholesterol levels were found in 5 patients. E.C.G changes

of previous M.I was observed in 11 patients (P= value 0.00). Cardiomegaly was seen in the chest x- ray among 5 patients. This association was statistically significant (P value .01). Echocardiographically LVSD was evident in all 12 patients with statistical significance (P= value 0.00). 7 C.A.D. patients had dense hemiplegia. One patient expired in the hospital.

SYMPTOMS OF C.A.D AND CARDIAC DYSFUNCTION

History suggestive of angina was observed in 29 patients (20%). H/O of palpitation was observed in 29 patients (20%) in the absence of valvular heart disease. Exertional breathlessness of varying degree was present in 49 patients. 4 out of 49 patients had shortness of breath at rest.



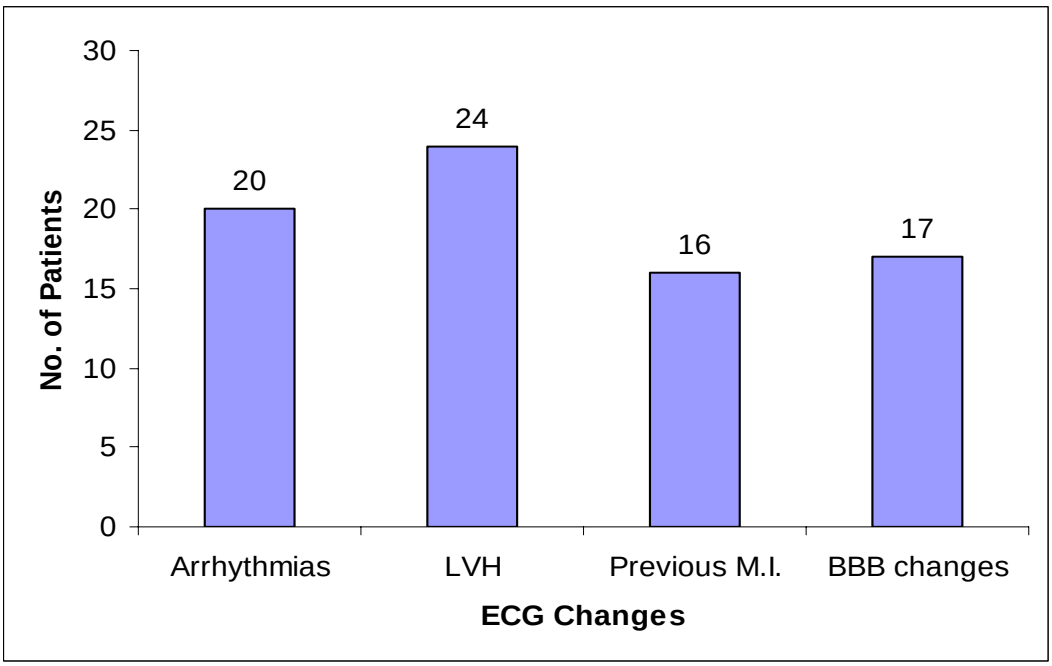
Symptoms of angina and palpitation were more frequently observed in 20 patients with hypercholesterolemia (P value .03). 14 out of 49 patients had chest x-ray changes of cardiomegaly. This was associated with shortness of breath N.Y.H.A class III and IV . This association had statistical significance (P value 0.00). E.C.G changes of previous M.I was present in 9 out of 49 patients. All of them had breathlessness at rest (P value 0.00). This association was statistically significant. There was no statistically significant association between symptoms of left heart failure with diabetes mellitus and hypertension. Left ventricular systolic dysfunction was evident echocardiographically in 9 patients with severe heart failure symptoms. This association was statistically significant (P= value 0.00). Past H/O stroke was observed in 6 patients. 7 patients had altered mentation (P = value 0.00).

11 patients had massive infarct on C.T. There was no statistically significant correlation between symptoms of left heart failure and mortality in the present study.

PAST HISTORY OF STROKE

Past history of stroke was present in 41 out of 142 patients. Out of which most of them were between 51 – 60 years of age. Men had more incidence of past H/O stroke men compared to women in ratio of 3:1. Out of 41 patients 16 had dense hemiplegia, 14 had altered mentation C.T changes of massive infarct was evident 23 patients. The severity of the stroke and clinical picture had significant statistical correlation with a P value of .00 .

ELECTROCARDIOGRAPHIC CHANGES



Electrocardiographic changes were observed in 76 out of 142 patients (53%). More men had E.C.G changes when compared to women in the ratio of 43:33. E.C.G changes of LVH was more frequently observed in 14 hypertensive patients followed by 10 smokers.

There was no significant association between H/O angina, palpitation with E.C.G changes. E.C.G. changes of previous M.I was observed in 11 out of 76 patients. All of them had shortness of breath on mild exertion and at rest. This association was statistically significant (P =value .00). 7 out of 11 patients with E.C.G changes of previous M.I had cardiomegaly on chest x-ray. This correlation had statistical significance(P= value .00). E.C.G changes of previous M.I was more frequently seen among the 11 out of 12

patients with past history of C.A.D followed by 6 diabetic patients. 19 out of 76 patients had dense hemiplegia. 11 patients with previous M.I changes had massive infarct on C.T . 4 out of 17 patients with B.B.B changes had diastolic dysfunction in the echocardiography. Another 4 patient with B.B.B changes had L.V systolic dysfunction 27 patients of varying E.C.G changes had L.V systolic dysfunction. 19 out of 24 patients with E.C.G changes of LVH echocardiograthically which was statistically significant (P =value .00). 9 out of 11 patients with ECG changes of previous MI had dense hemiplegia. This was also statistically significant (P = vlaue .00). 12 patients with changes of previous M.I had massive infarct on C.T (P value .00)

ECG changes of arrythmias was observed in 13 patients with hypercholesterolemia and 10 patients with hypertension. There was no statistical significance in the above association.

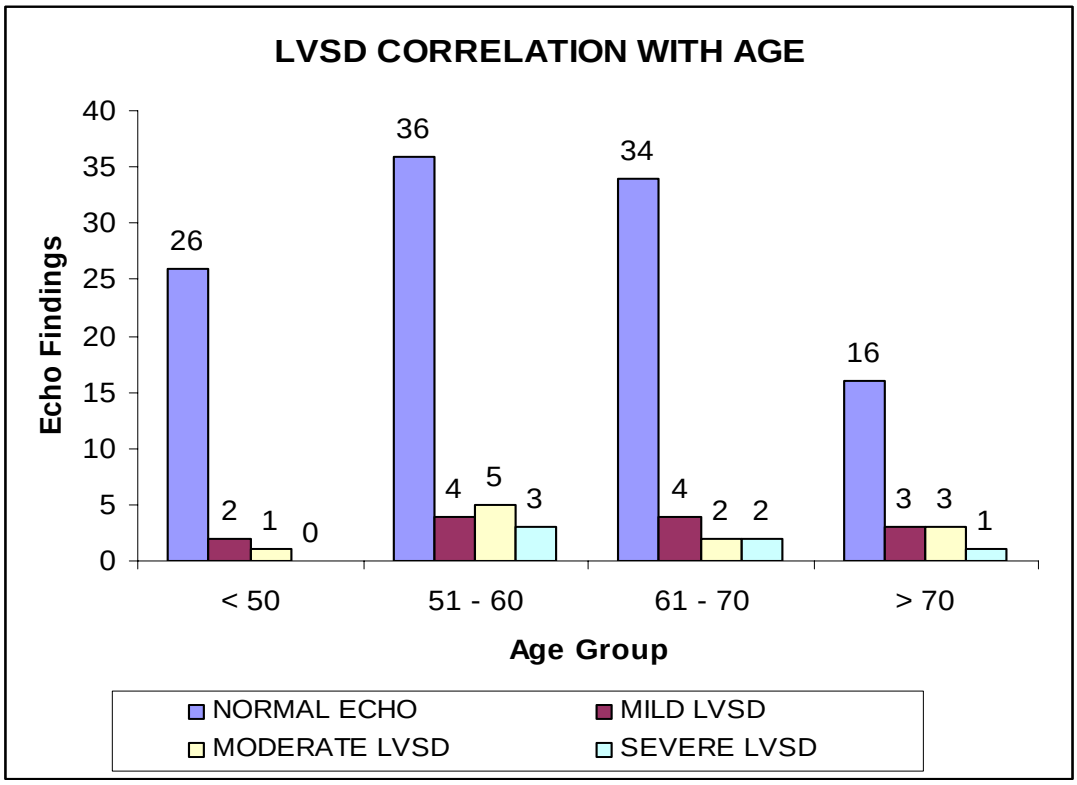
CHEST X-RAY CHANGES

28 patients had changes on chest skiagram. 5 patients had chest x- ray changes of pulmonary venous congestion. 23 patients had cardiomegaly on chest x – ray. Men and women showed chest x-ray changes in proportion of 16:12. 14 out of 23 patients with cardiomegaly on chest x- ray had N.Y.H.A class III and IV symptoms (P= value .00). All of the above patients had left ventricular systolic dysfunction which had no statistical significance. 3 patients had L.V diastolic dysfunction.

ECHOCARDIOGRAPHIC CHANGES

112 out of 142 patients had normal left ventricular systolic function echocardiographically.

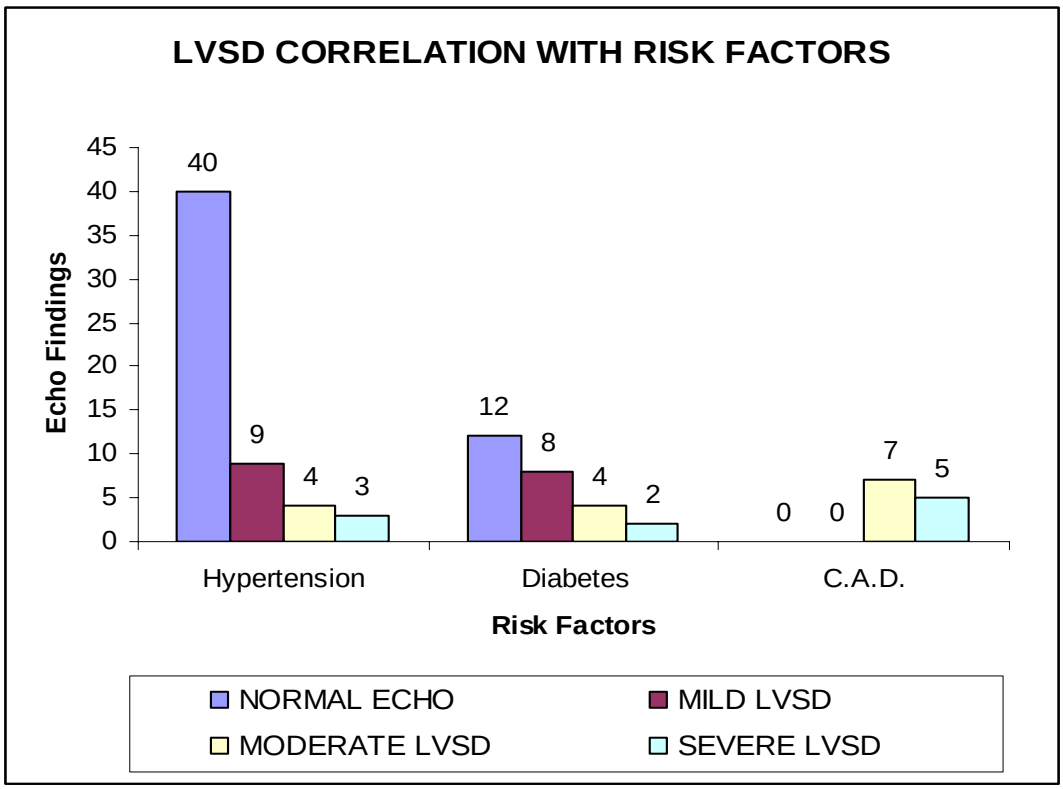
- 13 patients had mild left ventricular systolic dysfunction (9.15%).
- 11 patients had moderate left ventricular systolic dysfunction (7.75%).
- 6 patients had severe left ventricular systolic dysfunction (4.23%).



More number of patients were in the age group beteween 51-60 years followed by 61-70 years.

More men had left ventricular systolic dysfunction when compared to women in the ratio of 18:12.

16 hypertensives had left ventricular systolic dysfunction (P value = 0.12). 14 diabetic patients left ventricular systolic dysfunction which was statistically significant (P value .00). All the 12 C.A.D patients had moderate to severe left ventricular systolic dysfunction(P value = 0.00). 14 patients with N.Y.H.A class III symptoms had L.V.S.D (P value = .00).



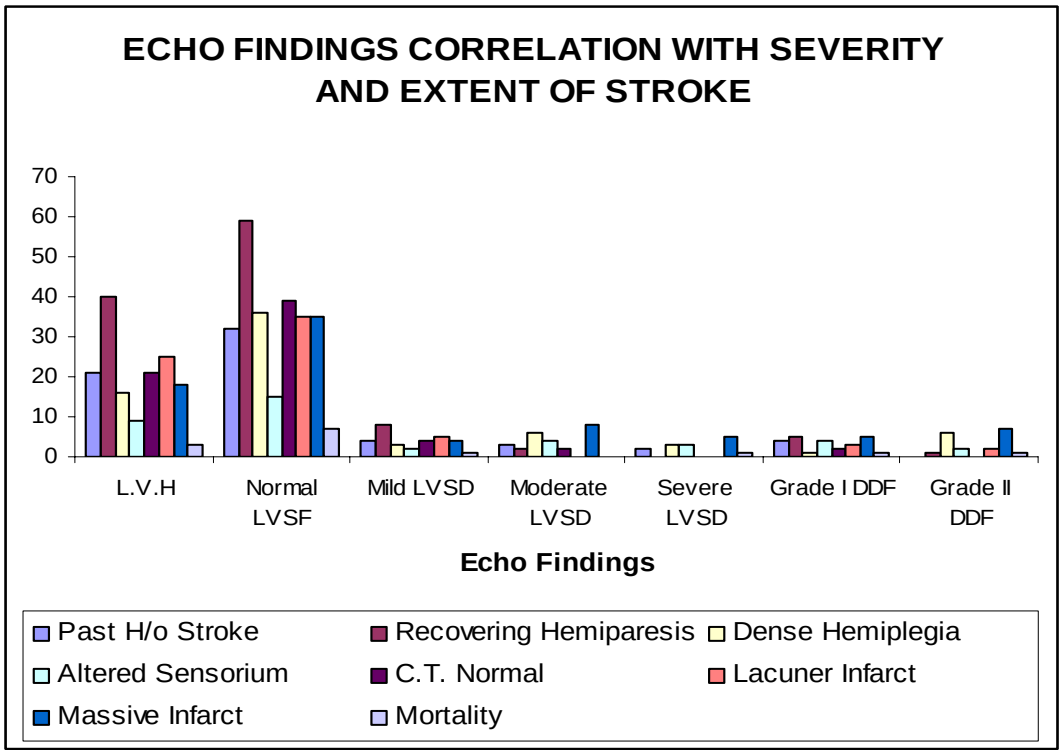
LEFT VENTRICULAR HYPERTROPHY

Left ventricular hypertrophy was observed in 65 patients. echocardiographically .Most of them were in the age group between 60-70 years followed by 50-60 years of age. More number of men had L.V.H when compared to women in the ratio of 41: 24 . L.V.H. was observed commonly in 39 smokers followed by 29 hypertensives. It was also evident in 13 alcoholics. 15 patients with L.V.H had history of angina and palpitation. NYHA class I symptoms where frequently observed in patients with L.V.H (P value = 0.09). 16 patients with LVH had dense hemiplegia (P value = 0.03). Massive infarct on C.T was evident in 18 patients (P value = 0.04). 6 patients had associated diastolic dysfunction echocardiographically. However this association was not statistically significant. 19 patients with E.C.G. changes of LVH had LVH echocardiographically. 11 patients with cardiomegaly on chest x-ray had L.V.H echocardiographically 9 patients with L.V.H had left ventricular systolic dysfunction echocardiographically . This association was statistically significant (P value= 0.02). 3 patients with L.V.H had in hospital mortality.

DIASTOLIC DYSFUNCTION

12 out of 142 patients had diastolic dysfunction echocardiographically 5 patients were in the age group between 51-60 years followed by 4 patients in the age group less than 50 years.

The number of men with diastolic dysfunction was more when compared to women in the ratio of 7:5.



There is no statistically significant association between diastolic dysfunction and history of angina, palpitation and symptoms of left heart failure 5 hypertensive patients had diastolic dysfunction. 2 diabetic patients had diastolic dysfunction. One patient with history of C.A.D had diastolic dysfunction. There was no statistically significant association between D.D.F and chest X- ray, E.C.G changes.

An attempt was made to correlate the clinical parameters of stroke – including severity of stroke, altered mentation, extent of stroke on C.T and mortality with L.V function. L.V diastolic dysfunction had statistically significant association with mortality when compared to L.V systolic dysfunction.

IN HOSPITAL MORTALITY

9 out of 142 patients had in hospital mortality. The average period of hospital stay was 12 days among this group. 3 were aged between 51 to 60 years and above 70 years. Death was observed more commonly in men (ratio of men: women being 5:4). Among those who expired, hypercholesterolemia was seen in 7 patients. Hypertension was observed in 5 patients. 2 patients had diabetes mellitus. One patient had coronary artery disease. 5 patients had symptoms of cardiac dysfunction on admission. This association between the symptoms of left heart failure and mortality had statistical significance (P value .00). cardiomegaly on chest skiagram was observed in 4 out of 9 patients. This association was statistically significant (P value .00).

Echocardiographic evidence of left ventricular dysfunction was observed in 4 patients. Out of which 2 patients had L.V. systolic dysfunction and 2 patients had L.V diastolic dysfunction. The association between L.V. diastolic dysfunction and mortality was statistically significant (P value 0.00).

DISCUSSION

The number of patients enrolled in the study was 142. V function studies in association with ischaemic stroke have been done by Allison G. Hays, M.D, Department of Medicine, New York (`1994-1997) in a subset of patients from the Northern Manhattan study 2006(NOMAS) [46].

In a study done by them, 270 patients of ischaemic stroke were evaluated for LV function. Framingham study and VH ef trial[51] analysed the various risk factors in association with LV dysfunction in ischaemic stroke patients.

E.C.G changes in stroke patients were analysed by DS. Goldstein[53] during 1979. In SOLVD[55] study, heart failure in stroke patients was evaluated.

AGE INCIDENCE

Among the patients studied 33% were between 51-60 years followed by 29% between 61-70 years. The average age of patients studied was 58years. The lesser incidence in people below 50 years of age could be because in our study we excluded patients with valvular heart disease and the causes of young stroke. The number of patients of over 70 years was also less is possibly because less no of people live beyond this age. The age incidence in the current study was similar to the northern manhattan study. In the NOMA study also the age of patients more than 70 and younger than 50 years was less.

SEX INCIDENCE

66% of patients in the present study were men as against 34% who were women. This is probably because smoking and intake of alcohol was observed in a majority of men in our group. This contrasts with the manhattan study figures (NOMA study) where 56% were women against 44% men.

The higher incidence of stroke in women of NOMA study could be due to the associated habits of smoking and alcohol intake in western women.

RISK FACTOR PROFILE

Smoking

51% of patients in current study were chronic smokers where as in NOMA study 23% were smokers. This difference could be due to the higher incidence of smoking in Indian men when compared to people of Western country where the habit of smoking was comparatively less than alcohol intake. The present study showed statistically significant association between smoking, hypertension and diabetes. The association between smoking and clinical severity and morbidity of the stroke was also statistically significant in the present study .

Alcohol

The incidence of alcohol intake was more in the NOMA study when compared to the present study. It was 40% in the NOMA study as compared

to the 23% of present study. This difference could be attributed to the higher incidence of alcohol intake in Western countries when compared to our country.

Hypercholesterolemia

The incidence of hypercholesterolemia was more in the present study (54%) where as it was less in the NOMA study (38%). The above difference could be due to the changes in the life style pattern of the people of our country with associated alcohol intake and smoking and also due to the increase in associated co morbid conditions like atherosclerosis, diabetes hypertension and C.A.D.

Hypertension

The incidence of hypertension was 39% in present study which was less when compared to NOMA study (i-e 78%). This could be due to the difference in the incidence of comorbid conditions like diabetes, C.A.D, dyslipidemia. The association of hypertension and C.A.D was statistically significant in the present study. The ECG changes of LVH and hypertension was statistically significant in the present study.

DIABETES MELLITUS

The incidence of diabetes in the present study was 18.3% as compared to the NOMA study of 43%. The above difference due to the sedentary life

style of the western people and also due to the higher incidence of obesity among the people of western countries . There was a statistically significant correlation between diabetes and LVSD. In hospital stay mortality was statistically significant among diabetic patients.

CORONARY ARTERY DISEASE

The incidence of C.A.D. was 8.4% in the present study as compared to the NOMA study of 31% the above difference could be due to the higher incidence of CAD in Western countries. Also this could be attributed to increase in associated comorbid conditions like diabetes, hypertension and dyslipidemia. The severity of the symptoms of left heart failure had statistically significant correlation with CAD in present study. ECG changes of M.I and CXR changes of cardiomegaly had significant correlation with CAD in present study. LVSD was more commonly observed in present study with statistical significance

SYMPTOMS OF CAD AND CARDIAC DYSFUNCTION

Since we were assessing the echocardiographic evidence of LV dysfunction, we evaluated the cardiac dysfunction in detail.

The symptoms of cardiac dysfunction was observed in the study group patients on admission. 20% had H/O angina and 49 patients had shortness of breath of varying degree. 4 patients had shortness of breath at rest. There was significant correlation between shortness of breath of severity and

E.C.G changes of previous M.I. Shortness of breath of at rest had a significant correlation with LVSD in present study. Clinical picture of stroke with altered mentation and severity of the stroke had statistically significant correlation with cardiac symptoms.

PAST HISTORY OF STROKE

Past history of stroke was obtained in all stroke patients in the present study. This was elicited to evaluate the significance of recurrent stroke in study group patients. H/O of recurrent of stroke was observed in more men when compared to women in the present study. This could be attributed to the difference in the number of men and women of study group. Associated smoking and alcohol habits were observed in men which was not observed in women . The severity of the stroke and clinical picture had significant statistical correlation with past H/O of stroke (P value = .00).

The recurrent stroke rate is much higher than the rate of first-ever stroke in patients with cardiac failure. Sacco et al[54]. found a 45% 5- year recurrent stroke rate in patients with cardiac failure.

ELECTROCARDIOGRAPHIC CHANGES

E.C.G changes in the stroke patients were compared with a study of E.C.G changes in stroke patients by DS Goldstein 1979[53] .

DS Goldstein study 1979 (Number of patients = 150)	Present Study ((Number of patients = 142)
Abnormal E.C.G (Total) 92%	53%
E.C.G. changes of arrhythmias (including tachycardia) 28%	14%
E.C.G. changes of L.V.H 26%	16.9%
E.C.G changes of -previous M.I 20%	11%
E.C.G changes of - B.B.B 20%	12%

The present study group patients had less frequency of E.C.G changes when compared to DS Goldstein study. However, the E.C.G changes of L.V.H and E.C.G changes of arrhythmias followed by E.C.G changes of previous M.I were commonly observed in both the studies . The above E.C.G. changes could be attributed to the underlying hypertensive, atherosclerotic cardiovascular disease, sympathetic hyperactivity, and possibly myocardial necrosis.

The current study showed that the E.C.G changes of previous M.I had a statistically significant correlation with severe heart failure symptoms of NYHA class III and IV (P value =.00). The above patients also had a statistically significant association with chest x – ray changes of cardiomegaly(p value =.00).

CHEST X-RAY CHANGES

Chest x- ray changes of cardiomegaly was observed in 23 patients. Out of the 23 patients 14 had NYHA class III and IV symptoms with statistical significance (P value = .00). The above patients also had left ventricular systolic dysfunction echocardiographically . However, this association was not significant statistically. 3 patients had diastolic dysfunction echocardiographically. This association had no statistical significance. The patients with chest x- ray changes of cardiomegaly had significant dense hemiplegia and massive infarct on C.T . This association between CXR changes of cardiomegaly with clinical severity and extent of stroke had statistical significance (P value .00).

16% of the patients had cardomegaly on CXR. Studies in ischaemic stroke patients on CXR findings are not much. However, in U.K T.I.A[52] study showed an association of 8% cardmegaly in T.I.A patients. The above difference could be attributed to the more incidence of cardiomegaly in completed stroke patients.

ECHOCARDIOGRAPHIC CHANGES

30 patients had left ventricular systolic dysfunction in the present study (21.13%) . The L.V.S.D changes of present study group was compared with L.V.S.D changes of NOMA study group as follows:

NOMA study (1994-1997) number of patients – 270	Present study number of patients -142
LVSD Mild 10.7%	9.1%
Moderate /severe 13.3%	11.98%
LVSD any level 24.1%	21.13%

LVSD of any degree was more frequent in stroke patients of the present study group. The incidence of LVSD was comparatively similar in both study groups. However, in the present study more men had LVSD than women in the ratio of 18:12. This association of higher incidence of LVSD in men was not statistically significant (P value = .13). In NOMA study, incidence in men and women was not much different (P value = .65).

LVSD was more common in the age group between 50-60 years followed by 60-70 years in the present study (P value 0.78). LVSD of any degree was significantly associated with ischaemic stroke among all age groups in NOMA study (P value 0.57).

16 hypertensives had L.V.S.D in the present study (P value= 0.12). 14 diabetic patients had LVSD with statistical significance (P value= 0.00) All the patients with E.C.G changes of previous M.I had LVSD of moderate to severe degree with statistical significance (Pvalue = 0.00). Clinical symptoms of severe heart failure (NYHA class III –IV) had a statistically significant

correlation with LVSD (P value 0.00).The patients with dense hemiplegia, altered sensorium associated with massive infarct on C.T had significant association with LVSD(P value = 0.00). LVSD had no statistically significant correlation with mortality in the present study.

In the survival and ventricular Enlargement (SAVE) study[56], patients with an EF of 29% to 35% had a stroke rate of 0.8% per year; the rate in patients with EF of 28% or less was 1.7% per year. There was an 18% increment in the risk of stroke for every 5% decline in EF.

DIASTOLIC DYSFUNCTION

12 patients had diastolic dysfunction in the present study. Most of them were between 51-60 years followed by less than 50 years. The number of men was more than women in the ratio of 7:5.

Diastolic dysfunction was more frequent in hypertensives than diabetics in the ratio of 5:2. One patient with C.A.D had diastolic dysfunction. There is no statistically significant association between history of angina, palpitation and symptoms of left heart failure and diastolic dysfunction. The association between D.D.F and chest x- ray, E.C.G changes is also statistically not significant. There is no significant association between diabetes, C.A.D and diastolic dysfunction in the present study. However, association between mortality and diastolic dysfunction was statistically significant (P value = 0.01).

IN HOSPITAL MORTALITY

9 patients had in hospital mortality in the present study. Mortality was more in men than women in the ratio of 5:4. 7 out of 9 patients had hypercholesterolemia. 5 patients had hypertension. Two patients had diabetes mellitus. One patient had coronary artery disease.

NYHA class I symptoms was observed in two patients on admission (P value =0.00). Cardiomagely on chest x- ray was observed in four patients. This association was statistically significant (P value= 0.00). The clinical picture and extent of stroke was also severe among the patients with diastolic dysfunction. Four patients had altered sensorium, 5 patients had massive infarct on C.T, one patient had dense hemiplegia (P value =0.04)

CONCLUSIONS

- LVSD was observed in 30 patients of the present study (21.13%).
- Diastolic dysfunction was observed among 12 patients (3.4%).
- Association of LVSD with clinical severity and extent of the stroke had of positive correlation statistically .
- Association of LVSD with in hospital stay mortality was not significant.
- Hypercholesterolemia was observed as the most common risk factor among the ischaemic stroke patients.
- Coexisting coronary artery disease and diabetes mellitus had positive correlation with left ventricular systolic dysfunction.
- Smoking was one of the most common risk factor observed among the ischaemic stroke patients.
- Patients with symptomatic heart failure of NYHA class III and IV had a positive correlation with left ventricular systolic dysfunction and clinical morbidity in ischaemic stroke.
- ECG changes of previous MI had statistically significant correlation with left ventricular systolic dysfunction and clinical morbidity in ischaemic stroke
- Chest x- ray changes of cardiomegaly was observed among 4 out of 9 patients with in hospital stay mortality.
- LVSD had no positive correlation with mortality .
- 6 out of 12 patients with diastolic dysfunction were asymptomatic.

PROFORMA

Name: Age: Sex:

D.O.A: D.O.D: I.P.NO: Ward

Complaints:

- Headache
- Vomiting
- Seizures
- Loss of consciousness
- Aphasia
- Sensory Disturbance
- Weakness of limbs
- Symptoms of cardiac dysfunction
- H/O angina
- H/O palpitation

Breathlessness on exertion

Past History:

- Diabetes
- Hypertension
- T.I.A
- H/O Cerebrovascular accident
- C.A.D

Personal History

- Alcohol Intake
- Smoking

Treatment History

General Examination

Pulse :

B.P. :

C.V.S :

C.N.S :

Higher Functions

Cranial Nerves

Motor

Sensory

Other Systems

Investigations

- R.F.T
- C.H.G
- Lipid Profile
- 12 lead E.C.G
- Chest X- ray
- Echocardiography
- C.T Brain

Mortality

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age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	
65	M	1	1									
70	M	1										
56	M	1	1	1		1						
75	M			1								
55	M	1	1									
40	M	1							1	1		
56	M	1										
70	M			1								
40	M	1	1	1								
56	M	1	1									
60	M	1										
40	M	1	1		1				1			
67	M	1	1		1			1	1	1		
63	M	1		1								
65	M	1		1								
50	M	1	1		1				1			
62	M	1										
67	M	1	1			1						1
40	M	1	1			1						1
55	M	1	1									
49	M	1		1								
64	M	1										
80	M	1	1					1				
40	M	1										
45	M	1	1									
41	M	1										
60	M	1										
50	M			1	1	1				1		
52	M											
52	M								1			
72	M											
42	M											
80	F							1		1		
70	F			1	1			1				
51	F											
75	F				1							
63	F			1	1			1				
48	F			1								
80	F								1			
68	F			1						1		
70	F			1						1		
56	F			1							1	
69	F			1							1	
45	F									1		
70	F			1						1		
80	F			1					1		1	
45	F			1				1	1	1		
68	F			1				1	1			
80	F			1				1	1		1	

65	F				1			1		1	
55	F			1			1				
50	F						1	1			
63	F									1	
65	F						1			1	
50	F			1	1						
65	F										
43	F			1							
50	F			1	1						
55	F			1							
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48	F			1							
55	F			1							1
75	F			1			1				
70	F									1	
55	F			1			1				
43	F			1							
58	F			1	1	1	1	1			1
70	F			1	1		1				
50	F			1							
45	F			1							
62	F			1	1						
54	F			1		1					
70	F			1							
66	F			1				1			
50	F			1		1		1			
65	F			1	1		1	1			
70	F			1	1	1			1		
65	F			1	1						
39	F			1							
57	F			1					1		
75	M			1					1		
75	M					1					1
60	M							1			
60	M	1					1				
90	M				1			1			
42	M	1					1				
64	M			1	1				1		
72	M	1	1							1	
84	M			1			1	1		1	
58	M			1					1		
52	M	1						1	1		
72	M	1									1
48	M	1	1								
55	M	1	1				1	1			
52	M	1					1		1		
65	M	1					1	1	1		
40	M	1	1								
62	M	1						1		1	
88	M	1	1				1	1			
45	M	1	1					1			

54	M	1	1				1								
48	M	1					1								
56	M	1						1							
75	M			1	1					1					
65	M	1					1	1				1			
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74	M	1	1												
52	M	1		1											
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82	M														
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78	M	1			1	1									
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80	M	1			1										1
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60	M	1	1	1	1										
64	M	1	1			1									
55	M	1		1											
60	M	1			1	1									1
70	M	1		1			1	1							
75	M	1					1								
62	M	1	1												
55	M			1	1		1	1							1

Smoke - Smoking, H.T - hypertension, D.M - Diabetes Mellitus, CAD - Coronary artery dis
CXR - Chest X Ray, ECG 1 - Normal, ECG 2 - Arrhythmia, ECG 3 - LVH, ECG 4 - Previou
Rec HP - Recovering hemiparesis, Dense HP - Dense hemiplegia, CT N - CT normal, Lac

NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5	CXR1	CXR2	L.V.H	ECHO-NSI	MILD
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	1			1						1	
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	1				1				1	1	
	1	1								1	
				1						1	
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	1	1							1	1	
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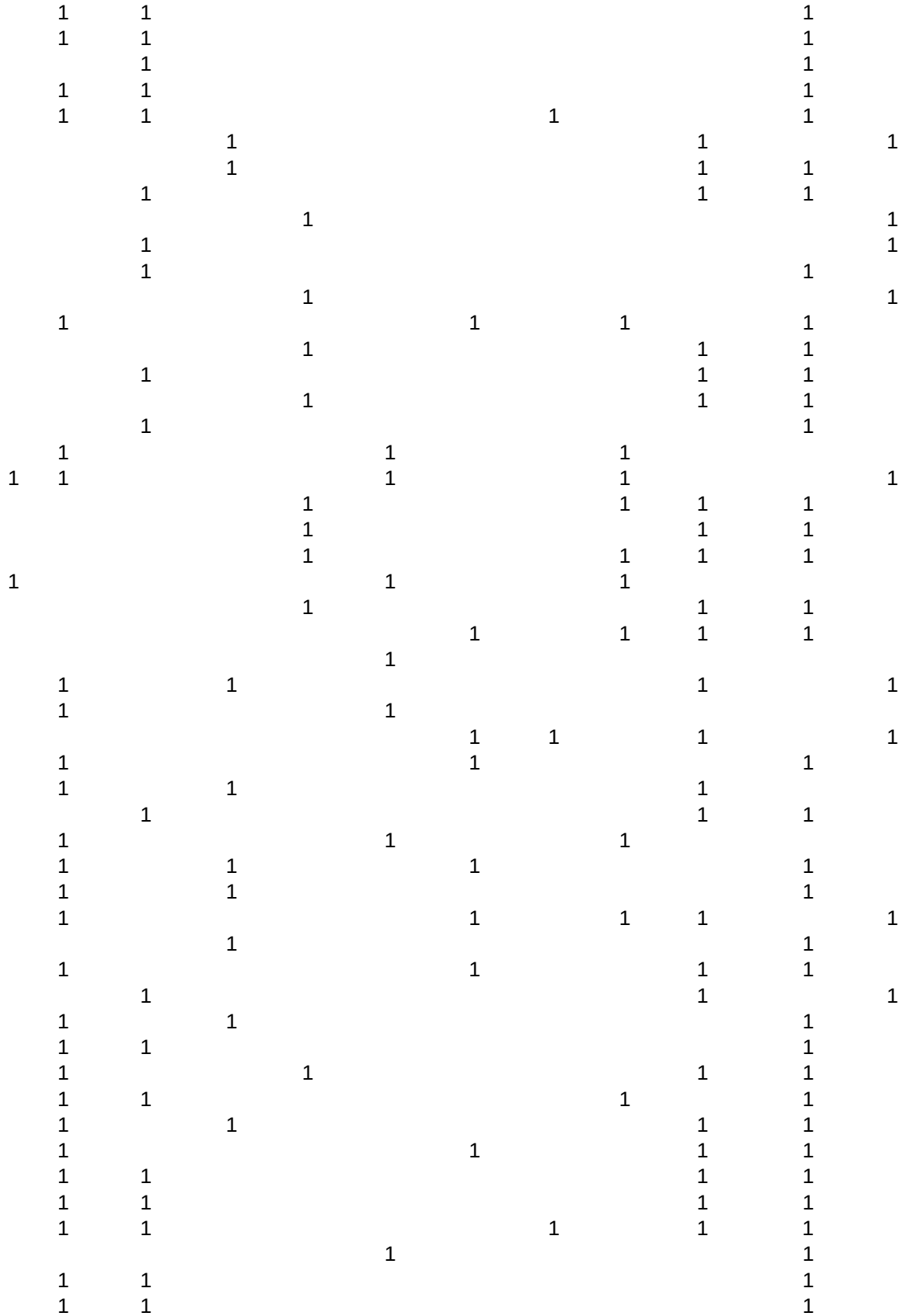

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ease, NYHA - New York Heart Association, H.C - Hyper cholesterolemia
us MI, ECG 5 - BBB, Mild - Mild LVSD, Moderate - Moderate LVSD, Severe - Severe LVSD, NS - Normal stu
:. Infr - Lacunar infarct, Mass. Infr - Massive Infarct, Mort - Mortality

MOD	SEVERE	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	
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S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
1	65	M	+	+										+		+			
2	70	M	+											+			+		
3	56	M	+	+	+		+						+				+		
4	75	M			+									+				+	
5	55	M	+	+										+	+				
6	40	M	+						1	1							+		
7	56	M	+											+			+		
8	70	M			1										1				
9	40	M	+	+	1									1	1				
10	56	M	+	+										1	1				
11	60	M	+												1				
12	40	M	+	+		1			1					1	1				
13	67	M	+			+		+	+	+				+			+		
14	63	M	+		+									+	+				
15	65	M	+		+										+				
16	50	M	+	+		+			+					+	+				
17	62	M	+												+				
18	67	M	+	+			+					+		+				+	
19	40	M	+	+			+					+						+	
20	55	M	+	+											+				
21	49	M	+		+									+		+			
22	64	M	+												+				

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
23	80	M	+	+				+						+	+				
24	40	M	+												+				
25	45	M	+	+										+	+				
26	41	M	+												+				
27	60	M	+													+			
28	50	M			+	+	+				+			+				+	
29	52	M												+	+				
30	52	M							+					+	+				
31	72	M												+	+				
32	42	M												+			+		
33	80	F						+		+				+				+	
34	70	F			+	+		+								+			
35	51	F														+			
36	75	F				+								+	+				
37	63	F			+	+		+									+		
38	48	F			+												+		
39	80	F							+					+	+				
40	68	F			+					+				+			+		
41	70	F			+					+				+		+			
42	56	F			+						+					+			
43	69	F			+						+			+		+			
44	45	F								+									+

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
45	70	F			+					+					+				
46	80	F			+				+		+			+					+
47	45	F			+			+	+	+				+	+				
48	68	F			+			+	+					+	+				
49	80	F			+			+	+		+								+
50	65	F				+			+		+			+	+				
51	55	F			+			+						+	+				
52	50	F						+	+						+				
53	63	F									+			+	+				
54	65	F						+			+			+	+				
55	50	F			+	+										+			
56	65	F														+			
57	43	F			+										+				
58	50	F			+	+											+		
59	55	F			+										+				
60	55	F				+									+				
61	48	F			+												+		
62	55	F			+							+		+					+
63	75	F			+			+									+		
64	70	F									+				+				
65	55	F			+			+									+		
66	43	F			+										+				

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
67	58	F			+	+	+	+	+			+		+				+	
68	70	F			+	+		+					+	+				+	
69	50	F			+												+		
70	45	F			+												+		
71	62	F			+	+											+		
72	54	F			+		+						+					+	
73	70	F			+												+		
74	66	F			+				+										+
75	50	F			+		+		+									+	
76	65	F			+	+		+	+					+		+			
77	70	F			+	+	+			+				+				+	
78	65	F			+	+													+
79	39	F			+									+					+
80	57	F			+					+				+		+			
81	75	M			+					+					+				
82	75	M					+					+		+				+	
83	60	M							+					+		+			+
84	60	M	+					+						+		+			
85	90	M				+			+					+					+
86	42	M	+					+								+			
87	64	M			+	+				+				+					+
88	72	M	+	+							+				+				

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
89	84	M			+			+	+		+			+		+			
90	58	M			+					+				+	+				
91	52	M	+						+	+				+			+		
92	72	M	+									+		+	+				
93	48	M	+	+										+		+			
94	55	M	+	+				+	+					+					+
95	52	M	+					+		+				+	+				
96	65	M	+					+	+	+				+	+				
97	40	M	+	+										+	+				
98	62	M	+						+		+							+	
99	88	M	+	+				+	+					+	+				
100	45	M	+	+					+					+	+				
101	54	M	+	+				+						+	+				
102	48	M	+					+						+	+				
103	56	M	+						+					+	+				
104	75	M			+	+				+				+	+				
105	65	M	+					+	+		+			+					+
106	50	M	+											+			+		
107	70	M	+									+		+		+			
108	63	M	+	+											+				
109	74	M	+	+											+				
110	52	M	+		+									+		+			

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
111	62	M	+	+							+					+			
112	70	M													+				
113	43	M													+				
114	51	M	+		+												+		
115	60	M	+	+										+					+
116	82	M												+					+
117	50	M	+									+		+				+	
118	59	M										+		+		+			
119	48	M	+	+											+				
120	42	M	+																+
121	45	M	+											+		+			
122	42	M		+										+		+			
123	66	M	+											+			+		
124	69	M	+		+	+						+							+
125	78	M	+			+	+											+	
126	53	M	+	+										+	+				
127	80	M	+									+			+				
128	66	M	+												+				
129	60	M	+	+											+				
130	40	M	+	+										+	+				
131	47	M	+											+	+				
132	80	M	+			+						+		+					+

S.No.	age	sex	smoke	alcohol	H.T	D.M	C.A.D	ANGINA	PALPIT	NYHA-1	NYHA-2	NYHA 3	NYHA 4	H.C	ECG1	ECG2	ECG3	ECG4	ECG5
133	60	M	+														+		
134	56	M	+	+													+		
135	60	M	+	+	+	+											+		
136	64	M	+	+			+						+					+	
137	55	M	+		+									+	+				
138	60	M	+			+	+					+						+	
139	70	M	+		+			+	+						+				
140	75	M	+					+						+					
141	62	M	+	+										+	+				
142	55	M			+	+		+	+			+			+				

Smoke - Smoking, H.T - hypertension, D.M - Diabetes Mellitus, CAD - Coronary artery disease,
NYHA - New York Heart Association, H.C - Hyper cholesterolemia ECG1 - Normal ECG 2 - Arrhythmia
ECG 3 - LVH ECG 4 - Previous MI ECG 5 - BBB

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
1	65	M			+	+								+			+			
2	70	M				+									+		+			
3	56	M							+				+		+					
4	75	M			+	+								+				+		
5	55	M				+								+			+			
6	40	M				+								+			+			
7	56	M			+	+									+			+		
8	70	M				+									+				+	
9	40	M			+	+							+	+			+			
10	56	M				+								+			+			
11	60	M				+								+				+		
12	40	M				+								+			+			
13	67	M			+	+								+			+			
14	63	M				+								+			+			
15	65	M				+									+		+			
16	50	M				+							+				+	+		
17	62	M				+								+						
18	67	M	+					+							+				+	
19	40	M						+								+				
20	55	M			+	+				+				+				+		
21	49	M			+	+							+		+		+			

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
22	64	M			+	+								+				+		
23	80	M				+								+			+			
24	40	M			+	+							+	+				+		
25	45	M				+								+						
26	41	M				+									+			+		
27	60	M			+	+							+		+					
28	50	M						+					+	+			+			
29	52	M				+								+			+			
30	52	M				+								+				+		
31	72	M				+							+		+				+	
32	42	M		+	+	+							+			+	+			
33	80	F			+	+									+				+	
34	70	F			+	+								+			+			
35	51	F			+	+								+			+			
36	75	F				+									+		+			
37	63	F			+	+								+			+			
38	48	F			+	+							+	+				+		
39	80	F				+								+			+			
40	68	F		+	+		+							+				+		
41	70	F		+	+	+									+		+			
42	56	F				+							+		+			+		
43	69	F			+	+								+				+		

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
44	45	F			+	+				+				+			+			
45	70	F		+	+	+								+			+			
46	80	F				+								+				+		
47	45	F				+									+				+	
48	68	F				+							+			+			+	
49	80	F			+	+							+	+			+			
50	65	F				+									+				+	
51	55	F				+								+			+			
52	50	F				+								+			+			
53	63	F				+									+		+			
54	65	F	+			+							+		+		+			
55	50	F			+		+							+			+			
56	65	F			+	+								+				+		
57	43	F			+	+								+				+		
58	50	F					+						+		+		+			
59	55	F					+							+				+		
60	55	F				+								+			+			
61	48	F					+							+			+			
62	55	F		+		+									+			+		+
63	75	F			+	+									+		+			
64	70	F			+	+								+			+			
65	55	F			+	+								+				+		+

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
66	43	F				+								+			+			
67	58	F		+				+		+				+			+			
68	70	F		+			+								+				+	+
69	50	F		+	+	+				+			+	+				+		
70	45	F			+	+								+				+		
71	62	F		+	+	+								+			+			
72	54	F		+					+						+				+	+
73	70	F			+	+								+				+		
74	66	F		+	+	+									+		+			
75	50	F						+							+				+	
76	65	F			+		+						+	+				+		
77	70	F						+							+				+	
78	65	F	+		+		+							+			+			
79	39	F				+				+				+				+		
80	57	F			+						+				+				+	
81	75	M			+	+							+		+				+	
82	75	M		+					+						+				+	
83	60	M				+										+			+	
84	60	M				+							+		+				+	
85	90	M		+	+		+							+				+		
86	42	M				+							+	+				+		
87	64	M			+	+								+				+		

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
88	72	M			+		+						+		+				+	
89	84	M				+							+		+				+	
90	58	M				+								+				+		
91	52	M			+	+								+			+			
92	72	M		+		+							+		+				+	+
93	48	M			+	+									+				+	+
94	55	M			+	+							+		+				+	
95	52	M			+	+								+			+			
96	65	M			+	+								+			+			
97	40	M	+		+	+				+			+		+				+	
98	62	M				+							+		+				+	
99	88	M				+							+		+				+	
100	45	M				+									+				+	
101	54	M				+							+			+			+	
102	48	M			+	+							+			+			+	
103	56	M			+	+								+				+		
104	75	M				+					+		+			+			+	+
105	65	M				+														
106	50	M		+	+	+				+			+			+			+	
107	70	M			+	+									+				+	+
108	63	M				+									+				+	
109	74	M				+										+			+	

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
110	52	M				+									+				+	
111	62	M	+			+								+			+			
112	70	M			+	+								+				+		
113	43	M				+								+			+			
114	51	M			+	+										+			+	
115	60	M				+									+				+	
116	82	M				+				+						+			+	+
117	50	M		+				+					+			+			+	
118	59	M		+				+					+			+			+	
119	48	M				+							+	+				+		
120	42	M			+	+				+			+			+			+	
121	45	M					+							+				+		
122	42	M				+								+				+		
123	66	M			+	+							+			+			+	
124	69	M		+					+							+			+	
125	78	M						+							+	+			+	
126	53	M				+				+						+			+	
127	80	M		+	+			+							+				+	
128	66	M			+	+								+				+		
129	60	M			+	+								+				+		
130	40	M			+	+									+				+	
131	47	M				+									+			+		

S.No.	age	sex	CXR1	CXR2	L.V.H	ECHO-NSF	MILD	MOD	Severe	DDF1	DDF2	DDF3	H/O Stroke	Rec. HP	Dense HP	Alt. Sens	CT - N	Lac. Infr	Mass Infr	MORT
132	80	M		+			+									+			+	
133	60	M			+	+								+				+		
134	56	M			+	+								+				+		
135	60	M			+		+						+			+			+	
136	64	M		+					+							+			+	
137	55	M			+	+							+			+			+	
138	60	M		+					+				+			+			+	
139	70	M			+	+								+				+		
140	75	M			+			+							+				+	
141	62	M			+	+							+	+				+		
142	55	M		+	+	+							+			+			+	

CXR1 - Pulmonary Venous congestion CXR2 – Cardiomegaly LVH – LVH in Echo Echo NSF – Normal Systolic Function

MILD – Mild LVSD MOD – Moderate LVSD Severe – Severe LVSD DDF - Diastolic Dysfunction

Rec HP - Recovering hemiparesis Dense HP - Dense hemiplegia CT N - CT normal Lac. Infr - Lacunar infarct

MASS. Infr - Massive Infarct MORT - Mortality